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DISEASES OF THE NERVOUS SYSTEM

A PRACTICAL JOURNAL ON PSYCHIATRY AND NEUROLOGY

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DISEASES of the NERVOUS SYSTEM

(PUBLISHED MONTHLY)

A PRACTICAL JOURNAL ON PSYCHIATRY AND NEUROLOGY

Nervous Complications in Infectious Diseases*

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Oftentimes nervous complications in common somatic maladies are encountered which may not be easily understood or diagnosed. They may be non-specific in nature, caused by the various infectious or toxic processes. Their anatomical basis may be some sort of a parenchymatous alteration of the nervous cells and other elements or some kind of circulatory disturbance. It would indeed be remarkable if the delicate structure of the central nervous system did not participate in the toxic alteration of the blood flow or of the permeability of the capillaries in infections and intoxications. The nervous tissue as well as the skin are ectodermal in origin. Since we are accustomed to see various skin reactions in many infectious and toxic diseases why should the nervous tissue be spared?

Organic damage will not necessarily produce the same clinical picture in all individuals, particularly with regard to vegetative responses. A person suffering from some instability of the autonomous nervous system, often described as vagotonia or sympathiotonia, will exhibit more vegetative symptoms than a more stable individual. The former may react excessively in temperature, loss of sleep, irritability, fatigue, pain, etc. This applies also to after effects from infectious diseases. Such effects are frequently observed during convalescence.

Almost every infectious disease may show neurological complications. Symptoms which indicate some affection, particularly of the brain, are characteristic in this group of diseases. Headache, general malaise, irritability, fatigue, disturbance of sleep, vegetative disorders such as palpitation, abnormal perspiration and fever are common and are manifestations of the participation of the brain in the disease process. A more severe degree of the affection of the central nervous system may be seen in the various states of impairment of the patient's consciousness which may increase gradually from an inconspicuous listlessness to stupor and coma. Muscular twitching is common especially in infections with high fever and grave toxemia. Epileptic seizures point to severe, probably more localized affection of the brain or have to be regarded as a reaction of a potential epileptic individual subjected to a diffuse brain alteration.

Participation of the meninges in infectious diseases is often observed. The cardinal symptoms are headache, stiffness of the neck, general hypersensitiveness, occasionally nausea and vomiting and the classical signs of Kernig and Brudzinski. Lobar pneumonia, particularly of the upper lobes, is frequently accompanied by such an irritation of the meninges. We refer here to a meningism or a serous meningitis, a syndrome which must be distinguished from a true infectious meningitis. The clinical symptoms may be ambiguous but a spinal puncture affords correct diagnosis, revealing in a case of meningism a clear sterile spinal fluid under increased pressure with only a negligible increase of cells, mostly lymphocytes, and a minor elevation of the protein content.

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In doubtful cases it is well to repeat the spinal puncture to be certain that the findings of a meningism were not the first stages of a true meningitis. Almost any bacterial infection, including influenza, numerous types of respiratory and intestinal infections, even gonorrhea, may lead to a true meningitis. Whereas no special therapy, aside from spinal puncture, is indicated in meningism, active therapy must be started immediately in cases of a true meningitis. This treatment, without going into detail, consists primarily of the use of sulfonamide drugs, which have proved effective in infections of the meninges. One of the most difficult forms of meningitis to treat is influenza meningitis, but even there the combination of a vigorous treatment with sulfadiazine and the administration of a specific serum directed against the protective capsules of the influenza bacillus, has greatly improved our therapeutic chances. Meningitis although conquered by treatment may leave residuals of which unilateral or bilateral deafness, chronic hydrocephalus, complex effects of chronic fibrous arachnoiditis may be mentioned. In some instances the clinical syndrome of a meningism may cover an encephalitic complication. Obviously a tuberculous meningitis, frequently insidious in its beginning stages, has to be ruled out.

The peripheral nerves often exhibit general tenderness, spontaneous pain and paraesthesias. The pain in the head and all over the body and the tenderness of muscles here and there (often called "rheumatism") may only be the manifestation of a diffuse neuralgia, the effect of an irritation of the spinal roots rather than of the peripheral nerves. Certain infections as influenza are characterized by such diffuse neuralgic pains. The majority of these neuralgias clear up with the infection itself without involvement of the peripheral nerves. The neuralgic phase is by no means a definite indication of more serious nerve lesions to follow. A true polyneuritis betraying grave anatomical lesions of the peripheral nerves is seldom seen in the initial phases of a general infection.

A *polyneuritis* may be a complication of widely different infectious diseases or intoxications. The clinical aspect may show distinct features, as, for instance, in diphtheria, but the symptom complex "polyneuritis" in general is suggestive only of some sort of an underlying infectious or toxic process, not specific with regard to any particular infection. The diagnosis polyneuritis is employed wherever the impaired function of a number of separate nerves indicates an underlying disease process. The word polyneuritis, meaning an inflammation of a number of nerves, should not make us forget that the anatomical process may indicate a degeneration rather than inflammation. Aside from a preceding infectious disease there is so little difference between an infectious type of polyneuritis and the polyneuritis well known in certain vitamin B deficiencies that the suggestion has been made that all types of polyneuritis may be due to a deficiency in the vitamin intake or absorption.

Influenza, diphtheria, typhoid and paratyphoid infections, dysentery, malaria, gonorrhea, sepsis, erysipelas, rheumatic fever, typhus, smallpox, chickenpox, infectious jaundice, scarlet fever, measles and tuberculosis may be complicated by polyneuritis. Focal infections are sometimes discovered to be the cause of a polyneuritis. The first polyneuritic symptoms often appear several weeks after the acute infection, though a slightly elevated body temperature, general malaise and pains in the extremities during this interval indicate the disease is still active. The more acute the initial infection has been, the more rapid will be the development of the polyneuritic syndrome. The neuralgic pains and paraesthesias, numbness or other sensory disturbances start usually in the distal parts of arms and legs, affecting both sides symmetrically. Soon a motor weakness in legs and feet is felt and examination will reveal a diminution or abolition of the tendon reflexes of the weakened muscles. The cranial nerves—mostly on one side—may be affected; also the facial nerve, the eye muscle nerves, the trigeminus, vagus and hypoglossus in diminishing frequency. The examination of the spinal fluid may reveal considerable increase in albumen but much less in cells.

Individual cases of polyneuritis may evidence different symptoms and follow a different course. The predominance of a disturbance of the deep sensibility leading to an ataxia especially in the legs combined with loss of reflexes, motor weakness and superficial hyperesthesia in both feet, creates a clinical picture similar to locomotor ataxia

and is known under the name of pseudotabes. Another syndrome, characteristic and dreaded because of its occasional bad prognosis, is known as Landry's ascending paralysis.

The combination of a true polyneuritis with a myelitis has been discovered in a number of cases. Death may occur because of respiratory failure, but most cases of polyneuritis offer a favorable prognosis though the time necessary for complete recovery is unpredictable. Treatment of polyneuritis offers somewhat better possibilities with the advent of vitamin B. It is advisable to give large doses and to add thiamine chloride, about 30 mg. or more intravenously, daily. Application of heat to the involved extremities in the form of external heat or short waves is helpful. Massage, passive and active exercise as well as orthopedic measures should be employed at the proper time in order to facilitate return of function and counteract possible contractures and deformities.

Infectious diseases of longer duration and of a more chronic type may produce a *neurasthenic syndrome*, characterized by a combination of fatigue, feeling of exhaustion, great irritability, moodiness, lack of concentration, loss of memory, headaches, hypersensitiveness to all sort of stimuli, increase of reflexes and various vegetative disorders. This neurasthenic syndrome must not be confused with a true constitutional neurasthenia. It usually responds well to rest and all the therapeutic measures which are commonly used to build up the patient's energy, to restore sleep, appetite and other body functions.

Psychoses due to infections and intoxications are by no means rare. They belong to the group of exogenic or symptomatic psychoses. The general practitioner sees them more frequently than the psychiatrist and he should be able to recognize them, though the specialist's cooperation will often be necessary for diagnosis and correct treatment. The personality structure of a patient plays a determining role in the psychotic reaction to infections and intoxications; so does the state of health of the central nervous system. Psychopaths and neurasthenics are predisposed to such psychotic reactions as are chronic alcoholics, individuals with other brain damage or those in a poor physical condition. Yet a certain number of previously normal individuals may become psychotic in the course of an acute feverish or other disease because of some diffuse alteration of brain function. Primary diseases of the brain—inflammatory, circulatory, traumatic and brain tumors—can produce identical psychotic reactions in addition, of course, to their own specific clinical pictures.

Although some predisposition to psychotic relation may be present, their particular etiology—their "trigger"—is the internal disease. For this reason the diagnostic ability of the attending physician is of as much value as the psychiatric knowledge of the consultant. A patient may develop a psychosis in the course of any of the exanthematous diseases of a septic or tuberculous infection, of a pneumonia or pleurisy, of a rheumatic fever or chorea minor, of an anemia or other blood disease, of a diabetes or other metabolic or endocrinic disturbances, of circulatory diseases, malignancy or any cachexia and of all sort of intoxications.

The clinical features of symptomatic psychoses are characterized by the fact that disturbances of consciousness are the nucleus or center of the disturbance. The formation of psychotic symptoms is generally linked to a drowsiness or a mental confusion. Impaired are the patient's attention, perception, his retention and recalling of recent impressions as well as his thinking which becomes dreamlike and incoherent. His affectivity is altered and primitive fears and anxiety may dominate his emotional reactions. The impairment of the patient's mental activity may affect his thinking in the categories of space and time. The patient becomes disoriented, unable to recognize his environment and the continuity of his conscious existence is broken. He cannot distinguish the origin of various stimuli; whether they originate from his actual environment, from dreams, illusions, delusions or hallucinations. The patient's utterances become incoherent, confused and jumbled. His hallucinations and illusions seem real in his own mind and he reacts toward them as to any sort of reality. He might behold them in a more passive or apathetic way, or act under their influence, struggle, fight or shrink away.

In some cases an unnatural euphoric and moderately agitated excitation prevails but more frequently misinterpretations, disagree-

able and frightening delusions, and terrifying illusions lead to a state of motor excitement characteristic of these psychoses. We say the patient is in a *febrile delirium*. The delirium may occur in all degrees of severity, from a soft muttering with restless movements of the hands (flocculation), as in terminal stages of meningitis or typhoid fever, to incoordinate motor activity and violent manic aggressive states in which the patient becomes a danger to others as well as to himself. Sudden change from one type of psychotic state to another occurs, sometimes with lucid intervals. Restlessness with hallucinations and delirious agitation will usually increase toward evening. Most of these patients suffer from insomnia. They resist food and drink and to feed them artificially is often impossible. Their body temperature may rise to over 105° F. Epileptic seizures may complicate the picture. The prognosis of such violent cases of delirium is grave and the patient may pass into severe exhaustion, coma and death. In general one is seldom wrong in regarding a symptomatic psychosis as a grave complication of an infectious disease. Whenever a psychosis continues beyond the duration of an infection or intoxication the question will have to be answered whether this is an exogenic or symptomatic psychosis and not an endogenic or genuine psychosis, usually of a schizophrenic type, precipitated by acute brain damage. This question can be answered adequately only by the psychiatrist.

For the treatment of symptomatic psychoses the first step is the treatment of the underlying infection or toxic condition. The severely agitated or delirious patient should, if he is transportable, be transferred to a hospital or institution. If the transfer is inadvisable or impossible, experienced nursing care and supervision should be provided. These patients often refuse food and do not empty their bladders. They have to be protected in every way. What they need most is rest and sleep and this may not be an easy task. One may use the most effective hypnotics, chloral hydrate, sodium amytal intravenously, scopolamine with small doses of morphine, etc., one may try cool packs or sponge baths, yet there will always be cases which fail to respond to sedative therapy.

There are a number of infectious diseases which produce characteristic neurological complications and we shall discuss them individually.

Nerve paralysis in *diphtheria*, where early and sufficient antitoxin treatment has not been administered, occurs in 20 per cent or more of cases and is still seen, in spite of optimal treatment, in about 10 per cent. Malignant or hemorrhagic types show an even higher percentage. Laryngeal diphtheria leads to nervous complications much less frequently than the ordinary pharyngeal type. The first nerve symptoms usually occur about 8 days after onset of the disease and offer the classical picture of a paralysis of the palate with the typical nasal voice and regurgitation of fluid through the nose. The side with the more severe diphtheric lesion shows greater paralysis. This observation, namely that the primary diphtheric paralysis depends on the site of the diphtheric infection, indicates that the accumulation of toxin in the infected tissue damages the nerves directly. This local neuritic phase is relatively harmless in itself but it is a warning of more unpleasant complications to follow. As the toxin enters the circulation, the nervous system in general may be affected. This polyneuritic phase may set in at any time between the second and seventh week, seldom later. Of all the symptoms, the paralysis of the ciliary muscles of both eyes serving accommodation (easily diagnosed from the patient's inability to read) is most common. Paralysis of the external eye muscles is more rare. Other cranial nerves may become paralyzed but only the vagus paralysis is a frequent symptom. This time, pharynx and vocal cords become more or less paralyzed on both sides. The patient may feel weak, particularly in the legs and examination reveals a loss of tendon reflexes. The weakness may increase and finally lead to a complete motor paralysis. It may remain localized in both legs or may spread over arms, trunk and neck and eventually paralyze the respiratory muscles, intercostal muscles, and diaphragm, and may finally kill the patient. The most frequent sensory symptoms are tingling and numbness in hands and feet, which may be followed by what is known as a pseudotabes diphtherica. The prognosis remains doubtful as long as paralyses progress. A vagus paralysis may have a very detrimental effect on the heart and contribute to toxic heart failure.

Nervous complications in *typhoid fever* are essential symptoms of this disease—the typical headache and the various degrees of impaired consciousness with confusion, delirious states and the many psychotic manifestations. The neurasthenic syndrome in convalescents after typhoid fever is frequent. Meningitic symptoms may be so pronounced that only the spinal puncture, revealing the presence of a meningeal irritation in absence of a meningeal infection, will exclude an acute infectious meningitis. In rare cases typhoid bacilli are found in the spinal fluid, occasionally but not always causing a purulent meningitis. Meningeal symptoms may also be caused by a spondylitis. Cases of typhoid osteomyelitis of a vertebral body are by no means rare. They may cause a local pachymeningitis and cases have been reported where an epidural abscess, leading to a compression syndrome of the spinal cord, was found. Typhoid bacillus metastases may occur throughout the whole nervous system. The combination of a thrombotic or embolic circulatory lesion with such a metastatic infection is probably the cause of abscess formation in the brain, sometimes seen after typhoid fever. More often encephalomyelitic foci are spread all over the central nervous system. Clinical symptoms vary according to the localization of the foci, spastic types of paralyses being the most frequent. However, cerebellar syndromes and symptoms of a more or less complete transverse myelitis may be seen. Frequently polyneuritis after typhoid, which affects the cranial nerves as well as the spinal nerves, may occur in combination with an encephalomyelitis. The clinical syndrome will then become very confusing. Finally, trophic disturbances after typhoid have to be mentioned, not only the well-known loss of hair, but also circulatory disturbances, hyperhidrosis, etc. The prognosis of metastatic CNS lesions is not bad, probably because the typhoid bacillus produces only limited tissue necrosis.

Nervous complications in *streptococcus* infections are frequent. Any type of a septicemia may show general symptoms of a cerebral involvement similar to those in typhoid fever. Here again, metastatic brain lesions occur. Numerous small infected emboli may reach the brain, particularly in endocarditis. The clinical syndrome is characteristic, being composed of diffuse encephalitic symptoms in combination with transient apoplectic pareses, aphasias, etc., leading to massive, lasting paralyses. The temporary quality of the focal cerebral symptoms may be easily misinterpreted as angio-spastic phenomena or the like. One should always suspect the presence of an endocarditis in an unidentified febrile condition revealing the brain symptoms just mentioned. Brain complications may be seen in subacute, as well as ulcerative endocarditis. They may overshadow all other symptoms to such a degree that the diagnosis of the underlying pathology becomes difficult. The autopsy usually reveals infected embolic brain lesions and occasionally a massive brain hemorrhage on the basis of a ruptured mycotic arterial aneurysm. Brain abscesses may also be found. Sometimes the infection is localized in the meninges and may produce a purulent meningitis, not infrequently with hemorrhages in the subarachnoid space. Streptococcus infections of the skin, particularly erysipelas of the face, may spread into the brain. Here the CNS infection is apt to occur as a result of continuous infection of the veins and lymph vessels of the head. The situation resembles that in purulent infections of the ear and the sinus. A purulent meningitis with or without an infected sinus thrombosis is not a rare complication. Treatment and still more important, prophylaxis of these nervous complications in streptococcus, as well as in other bacterial infections, has made much progress through the use of sulfa drugs and will probably benefit even more by the administration of penicillin which has already proved its worth in cases of subacute endocarditis.

Acute rheumatic fever, regarded as a special manifestation of a streptococcus infection, can produce cerebral symptoms by way of embolic lesions originating in the heart in cases with endocarditis or myocarditis with heart failure. But aside from that the CNS may act in a specific rheumatic manner, by producing a pathognomic tissue reaction to the antigen. Such is apparently the pathology of St. Vitus dance or Sydenham's chorea. The meninges may react as an outspoken meningism, a syndrome described as "brain rheumatism." A spinal puncture in such cases reveals an increase in pressure but only moderate deviations from normal in cell count and albumen. The patient may be benefited

by the use of artificial fever, but this should be left to experienced specialists.

Scarlet fever is now recognized as an infection with a hemolytic streptococci. Ordinary scarlet fever may be complicated by nervous symptoms in early stages or after an interval of about 14 days. Many symptoms diagnosed as nervous scarlet manifestations are in reality caused by other scarlet complications, particularly otitis media. General brain symptoms with or without meningism in the first stage of the infection are common. Small children often develop convulsions during this soporous or delirious phase. Most severe is the involvement of the CNS in those dreaded fulminating toxic types which usually die within 48 to 72 hours, showing temperature rise sometimes to 110° F. A true scarlatinous meningoencephalitis, independent of other purulent complications, is rare. Once in a while one sees a hemiplegia, a cerebellar syndrome or extrapyramidal picture with chorea or athetosis which can only be explained as true scarlet fever complications. A neuritis or polyneuritis after scarlet fever occurs more often and the cochlear nerve may participate in such toxic nerve damage. Deafness without otitis media or labyrinthitis is explained by such a neuritis.

Nervous complications in virus diseases have been noticed for many years, but only since our knowledge of virus infections of the brain was advanced extensively by the clinical observations in the last period of epidemic encephalitis, and by the experimental work on acute poliomyelitis, have we become fully aware of the participation of the nervous system in virus diseases in general. In spite of the work accomplished, much research still remains to be done. What really happens in the CNS in measles, smallpox, chickenpox, German measles, mumps, and other virus diseases? The question whether CNS complications are the effect of the same virus active in the particular virus disease or of an activation of some unknown virus has not been definitely answered. The possibility that the CNS lesions might be a toxic manifestation of an allergic character is still under discussion.

Encephalitis in *measles* occurs in less than 10 per cent of cases. However, incidence is higher if only severe cases of measles are considered. General brain symptoms in the initial phase of the disease accompanied by high temperatures are not rare though convulsions seldom occur. A serous aseptic meningitis is seen occasionally and may be combined with oculomotor or abducens paralysis. More serious CNS symptoms may appear in the stage of defervescence when the eruption is disappearing or has already disappeared, but some instances are known in which a hemiplegia and other brain symptoms have developed during the first week. The onset of nervous manifestations may be accompanied by recurrence of fever and impairment of consciousness and at this time convulsions also may occur. General brain symptoms with or without fever may persist for days or weeks to be followed by focal brain symptoms. These symptoms show all the variety ascribed to brain lesion caused by disseminated encephalitis. The multiplicity of symptoms and symptom combinations reminds one of those in multiple or disseminated sclerosis. Yet there is hardly any doubt among neurologists and neuropathologists that this disseminated encephalitis has nothing to do with multiple sclerosis. Aside from pyramidal symptoms, one sees aphasias, dysarthrias, cerebellar disturbances, extrapyramidal manifestations like chorea and athetosis, spinal paralyses, even total transverse spinal lesions and neuritis optica. Again neuritic and polyneuritic symptoms may complicate the picture. Prognosis, in spite of the clinical picture, is not bad, although very severe comatous cases may succumb and other cases may remain paralyzed for life. There is no specific therapy any more than for acute poliomyelitis or other virus diseases.

Smallpox is so rare in this country that few men have had much personal experience with nervous complications in this disease. According to the literature, the onset of nervous symptoms seems to occur during the week following the eruption, though the symptoms may antedate the eruption or begin 2 to 3 weeks after the eruption has appeared. Clinical manifestations and pathology resemble those in measles.

Since 1922, in Europe and in this country, a number of cases of encephalitis have been observed after vaccination. The incidence rate of nervous symptoms following vaccination varies widely (between 1 in 5,000 in the spring of 1925 in the Low Countries and

1 in 100,000 in Germany, 1927-33). Kinnier Wilson has stated: "In view of the millions of vaccinations conducted yearly all over the world, the figures are in reality trifling." The critical period after vaccination seems to be around the 11th or 12th day. Symptoms, if any, usually develop rapidly with fever, drowsiness, stupor and often convulsions. Ocular palsies and trismus (as in tetanus, Hassin and Geiger) have been reported. The older the patient, the more frequently pyramidal lesions seem to dominate the picture. After about a week's duration, benign cases show improvement, followed by recovery. However, residual nervous and mental disturbances and epileptic seizures may remain. Mortality in some groups of severe cases has been about 50 per cent. Pathology is again similar to that in measles.

Chickenpox rarely develops nervous complications and then usually on the 5th day after the rash has appeared. In combination with more or less violent general brain symptoms, signs of focal lesions in the cerebrum, cerebellum and spinal cord often with meningitic symptoms appear. An interesting point with regard to meningoencephalitis in chickenpox is the relation of the chickenpox virus to that of herpes zoster, which Wallgren and others declare to be identical. The character of the lesions found in the white matter is similar to that in measles.

Encephalitis in German measles has been reported and is supposed to occur about 3 to 6 days after appearance of the rash. Most of these cases showed such pronounced inflammatory symptoms in the spinal fluid that the question arises whether the nervous symptoms were not the effect of complicating infections.

Parotitis epidemica or mumps fairly often shows nervous complications particularly meningitic symptoms. The spinal fluid may show increase of cells, even in the absence of clinical signs of meningitic irritation. Symptoms usually appear within the first week. During epidemics, cases of benign meningeal irritation without parotitis have been observed among soldiers exposed to an infection of mumps and it is not unlikely that some of those enigmatic cases of lymphocytic meningitis which one sees occasionally may be abortive forms of mumps. Differential diagnosis of a tuberculous meningitis may be difficult in the initial stages. Encephalitic syndromes, sometimes with convulsions and various cerebral, cerebellar and spinal symptoms, also cranial nerve symptoms, seem to be rarer than the polyneuritic variety which has been found at times to bear the characteristics of a meningo-radiculitis. Prognosis in general is favorable.

Nervous complications in *whooping cough* deserve a place for themselves. The nervous element in this disease has been recognized as an essential part of the clinical syndrome by outstanding clinicians and many claim that a spasmophilic diathesis is the decisive factor in the violent paroxysmal attacks of coughing as well as in the eclamptic fits so typical of complicated whooping cough. It remains doubtful whether a picture of disseminated encephalomyelitis occurs in whooping cough, although it has been found on autopsy that vascular lesions are the predominant findings in the brains of children who have died of nervous complications. These vascular lesions are similar in many respects to those seen in true spasmophilia. Here a prolonged spasm of the larynx and respiratory musculature leads to severe cyanosis and asphyxia and severe stasis within the cerebral vessels. If the spasm is not interrupted in time, permanent circulatory disturbances in the brain will follow. Children under 4 or 5 years and particularly under 2 years are subjected to severe spasmophilia as well as to convulsive seizures in whooping cough. Other lesions in the brain with less evidence of a veinous stasis remind one of those brain lesions seen in air or gas embolism and the conclusion offers itself that under the strain and abnormal state of the respiratory muscles, air may enter pulmonary blood vessels and from there reach the brain to cause numerous ischemic lesions. The clinical syndrome may be grave and the prognosis becomes bad once the eclamptic attacks terminate in coma. Death may ensue in one or a few days. In a number of cases the child survives, but will show nervous residua of the many possible brain lesions which have occurred.

8 South Michigan Avenue.

The Social Service Department in a Mental Hospital*

AVANELLE S. WELDON
Fort Worth, Texas

Psychiatric social work is no longer a lusty infant screaming for attention, nor yet is it a gawky adolescent whose motives and behavior are uncertain and unpredictable. It is, rather, a mature individual whose purposive, well-integrated demeanor has won for it a definite place among those undertakings designed to alleviate human distress. Psychiatric social work has been described as a "new emphasis rather than a new function." Dr. Adolph Meyer has stated that "Before our present-day type of social work in hospitals was organized, psychiatry here and there had for a century or so done a kind of social work under the name of 'after care.'" In Switzerland, France and many other countries a schedule of care was provided for patients discharged from the mental hospital. In the United States as far back as 1860 reports of some state hospitals indicate that the need for consideration of the patients' social problems at the time of discharge was recognized. In fact oftentimes the social investigation was done by the doctors themselves. More general interest in social readjustment was evidenced in 1894 when the American Neurological Association sent a letter upon the subject to prominent neurologists. Of the fifty replies received the majority expressed their decided belief that there were great advantages likely to result from "after care" work.

Our modern variety of psychiatric social work is truly therefore a new emphasis rather than a new function. Early in the twentieth century there appeared within psychiatry a shift of interest from diagnosis and classification of mental diseases to the personality of the patient in relation to his environmental situation. This brought about, on the part of social work, more attention to the psychological and mental factors in social maladjustment. Psychiatry turned to social work for help in the study of the personality in its social setting, just as social work turned to psychiatry for help in understanding baffling problems in attitudes and behavior.

In April, 1906, the first "after care" agent, a graduate of the New York School of Philanthropy, now the New York School of Social Work, was employed by the New York State Charities Aid. Five years later, an after care worker was appointed by the State Commission on Lunacy and assigned to Manhattan State Hospital. Also in 1906, Louisa Schuyler, having made an investigation of the After Care Societies in England, presented to the New York Commission on Lunacy a practical plan. Thus the interest in the social adjustment of patients discharged from state hospitals crystalized into a definite program.

It was Miss Mary Jarrett, who in 1913, at the Boston Psychopathic Hospital, outlined carefully a program of social work with mental patients. This has served more or less as a pattern, but the social service program of progressive hospitals is not static. It is readily changed and expanded to meet the needs of the community. It not only serves the patient, and his relatives, but it may play a major role in the all important matter of interpretation and preventive medicine. It is impossible to estimate the value in dollars and cents of a well-organized psychiatric social service department, for because of it, taxpayers pay for fewer days of hospital care for a great many patients. Numbers of out-patients may be helped to a satisfactory adjustment without ever having a day's hospitalization, and in this day when mental hospitals' admission rates are increasing alarmingly, these monetary savings are of prime importance.

Dr. William A. White in his book "Forty Years of Psychiatry," quoting statisticians, writes that in 1970, if the rate of admissions to mental hospitals continues as it has in the past, there will be about 950,000 residents of the United States who will be patients in mental institutions. This is more than double the number now cared for. Figures and tendencies shown by them indicate that mental disease is more prevalent than supposed, for it can hardly be true that these figures represent an increase in these disorders. Statistics for mental disease go back only to 1880 and in this brief period the statistical increase has been enormous. What is probably true is that mental disease is being discovered. The institutions are gaining the confidence of the

*Read at meeting of Texas Mental Hygiene Association, Houston, Texas, March, 1944.

public, and their help is being sought where before they were avoided. The understanding and concept of mental disease is being liberalized to include conditions which in the past were not accepted as such. The signs are clear that as much help as possible must be secured in handling the problem. Since there are not enough psychiatrists the psychiatric social worker specially trained and equipped may be part of the answer.

While there are many activities common to all social service programs, there is much latitude in selection of activities and application of policies. There are many hospitals whose programs are still centered narrowly around the patients who are committed, permitting limited contact with the community and with meagre provision for preventive or educational work. At the other extreme, there are state hospitals likewise concerned with treatment of patients, but moving forward with a growing community program which includes supervision of the patient after he leaves the hospital; establishment of preventive work and cooperative relationships with agencies concerned with mental hygiene.

In the first main division of activity, work with patients and families, the social service should be continuous, beginning in a contact with the relative bringing the patient to the hospital. In this initial interview many things can be accomplished. The relatives can be given some understanding of the hospital routine and function. A great deal of anxiety, guilt, humiliation, and confusion may be eased and the family may thus be released to form a more helpful working relationship with the doctor at the hospital. This contact is the first step in the process of social treatment, which later is to involve explanation to the family, further understanding of the family situation, preparation for the return of the patient, and follow-up after discharge. It may give the family a feeling that they know personally within the hospital some individual upon whom they can call.

There are some physicians who prefer to obtain the social history for themselves but for the most part they have found the social worker a capable ally in this, and there are several advantages which are the natural result of her securing the material. Usually, a more complete picture of the patient can be constructed if one can see him in his native setting. This would be too time consuming for the physician, but the home visit made by the social worker can be fitted into the schedule of each worker. Her observation and impressions of the patient's social setting are of almost as much importance as the factual material she is able to obtain from the family. It is not unusual that these impressions accurately reported furnish the only real clue to the patient's disorder. Then, too, because of her knowledge of the function of other social agencies she is able to determine the significance of their contact with the family and can secure their reports. If another social agency is already interested, she can help maintain this during the patient's hospitalization.

From a purely practical standpoint, the social worker can, as a result of her initial contact with the relatives, save many precious hours for the already over-worked doctor. Those of us who work in hospitals are all too familiar with the insistent demands relatives make upon the physician's time. Often enough the questions and problems they present can only be handled by the medical staff, but many inquiries can be taken care of by the social worker familiar with routine, procedure and such.

The amount and kind of service the social worker gives directly to the patient during hospitalization varies greatly. It, as all other functions of the social worker, should be planned in close cooperation with the medical staff, and certainly depends upon the circumstances in each case. The important thing is to see that the necessary treatment is given.

Many hospitals have found it advantageous for the social worker to be present at ward rounds, to be introduced to the new patient and later to become acquainted with him. In this way the worker can be identified with the hospital, yet, with her contact in the community can help the patient feel less isolated and provide a stimulus for his return to the community. Occasionally, patients are afraid of doctors but are willing to talk with a social worker whose approach is on an informal, friendly basis. Often a social worker can establish a helpful relationship with such patients by seeing that minor requests receive attention, and that social matters immensely important to the patient are handled. It is possible for the worker to interpret hospital

regulations and restrictions for the patient, decreasing his antagonism and resentment, and causing him to respond more readily to treatment procedures. Because of a supportive relationship the social worker can often help a patient span the gap between complete dependence while in the hospital and the independence he must assume upon his return to the community.

With the return of the patient to the community, the social worker's task is to aid him in his relationships to his home, friends and employers. This is known as family care or boarding home care. While it is not new, having been most successfully used in several European countries, it certainly has not been widely adopted in the United States. However, some state hospitals, notably those in Worcester, Massachusetts; Howard, Rhode Island; Poughkeepsie, New York, and others, have well-established programs. These are made possible by the skill of the social worker. Her role in liason between the hospital, patient and community is most important. She not only supervises the patients placed in homes but it is her task, as well, to secure and carefully select homes into which various types of patients can be advantageously placed. This means that the social worker must know and understand the needs of the patients referred to her, can interpret their problems in such a way that the interest of the community is stimulated and the boarding home keeper is helped to give the type of care desired.

From the viewpoint of the administrator this provides a way in which the population of the hospital can be decreased, representing a saving in dollars and cents. Individuals who have no homes to go to, or those who, for one reason or another, cannot be cared for by their families, yet who do not need the intensive care of the hospital, make up a large proportion of the population of the average state hospital. Many of these can make a most satisfactory adjustment in a proper boarding home where the per capita cost is considerably below that for care in the hospital. In addition, much is gained in human happiness, for most people are more comfortable and contented in the surroundings of a home. This type of care can also serve as the intermediate step between the protection afforded by a hospital and the sudden return to responsibility a patient faces when he is discharged from the hospital. When this transition is gradually made the rate of readmission has been found to be definitely lowered.

Many hospitals have extended their facilities to reach into the community and serve those individuals who do not require hospitalization but who are recognized as being in need of psychiatric attention. Usually, these out-patient clinics serve a dual purpose in that they also provide a means of continued supervision of patients released into the community on trial visits or parole. Cooperative relationships between the hospital and the social agencies of the community are of a great deal of importance. Often those individuals who come to the attention of society as needing psychiatric care are the same individuals who have had contact with various social and corrective agencies, and social workers have recognized their behavior as deviating from normal to the extent that skilled attention is necessary.

A clinic staffed by a psychiatrist, a psychologist and a social worker is the typical organization. In many of these clinics the social worker has been delegated the major responsibility for both a modified psychiatric and social treatment with the psychiatrist acting as supervisor and consultant. This task may be one of helping the worker who refers the patient to the clinic to understand the nature of the difficulty, to discuss the recommendations of the psychiatrist, to act as coordinator between the psychiatric treatment carried on by the clinic and the social treatment for which she herself is not directly responsible. Many social workers have proved themselves capable of assuming a large share of responsibility for the continued psychiatric treatment of paroled and discharged patients. A well-equipped worker with mature judgment and skill in handling problems inevitably will become an increasingly important member of the treatment team for there will never be enough psychiatrists to handle adequately the treatment of all emotional problems. The work of the psychiatrist and the social worker should be closely inter-related, each recognizing the area of treatment for which his skills and training fits him best, yet using to the fullest the special contribution each has to offer.

In many communities the feeling still exists that the mental hospital located a few miles out of town is not something one points to with pride and enthusiasm as a constructive agent, yet within that hospital are infinite resources for education and better living. Some communities are taking advantage of the skill and knowledge of hospital staff members by calling upon them to discuss emotional problems and mental disease. A social worker with her contacts with the various agencies in the locale can help to build up a friendly relationship between the hospital and the community that will be immensely beneficial to both. With the social worker constantly in touch with organizations and individuals the program of placing the released hospital patients may find a more cordial welcome. All too prevalent is the feeling that one who has had a mental or nervous disorder is to be shunned and regarded with fear and suspicion. Families themselves feel that they should keep secret the fact that a member of their group has had a period of hospitalization. This tends to impede the patient's readjustment in the community. While some former patients of mental hospitals have been able to make an adjustment in a community where they were not known it also has been demonstrated that an understanding family can materially assist the patient in his return to civil life.

A typical example of what can be done along these lines is the program of the Rhode Island State Hospital at Howard, Rhode Island. True the total area served by the hospital is small and almost everyone knows an individual who has at one time been a patient at the hospital. Nevertheless, the friendly, helpful interest of the community has definitely been increased through the program carried on by the Social Service Department. In addition to the work within the hospital, the physicians expect the social workers to make careful pre-parole plans for patients being released. They are expected to know and understand the patients' problems, the recommendations of the psychiatrist, the attitudes and environmental situation of the family, and to possess the knowledge of community resources which can be used to ease the way for the patient's return. These community contacts have penetrated every nook and corner; from the farmer in the rural area, to the housewife in the small town, to the busy bank president. While the origin of these contacts is not definitely known, they grew amazingly through the 1930's when jobs were hard to find and it was then that the medical staff felt that many a patient's release depended upon his or her ability to find a place in the community. The social workers accepted this as a challenge to their knowledge and to their skill in interpretation. First, one or two farmers agreed to try patients on their farms to see if they could work satisfactorily. Care was made in placing these people and the social worker gave close supervision and help to both patient and employer in working out differences. The word spread. A few more were placed. After a time the farmers' organization, the Grange, became interested, and its president and members began to look to the hospital as a source of reliable help.

A similar situation developed among housewives who were willing to accept patients within their homes as housekeepers when they knew that a social worker would be on hand to help them understand the behavior and needs of the individuals. Many patients were placed in industrial and business establishments, and throughout the state social workers knew the key individuals. There developed the friendliest of relationships between the hospital and all types of civic organizations. Members of the hospital medical and social service staffs were popular speakers at community functions and were often called upon to interpret emotional and mental disturbances and to suggest ways in which preventive measures might be taken. Thus the State Hospital for Mental Diseases became a community organization contributing much to the health, happiness and well-being of the community.

Now and in the near future when an ever-increasing number of young men and women who have suffered mental and emotional disorders are being discharged from the Armed Forces to the communities, it is of inestimable value to society that the already existing organizations dealing with these disturbances be prepared to interpret them to the community.

Treatment of Neuroses and Mental and Psychic Disturbances of War and Their Prognosis

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1. *Treatment for Combat Fear.*

Early heroic treatment has been found successful with many mentally maimed soldiers and sailors. The first and foremost requirement in treatment is absolute rest amid pleasant surroundings. Wholesome amusement, relaxing exercise, good palatable food and free discussion of the entire situation by the physician help to alleviate the condition. If the problem of sacrifice in relation to combat duty is understandingly discussed and objectively appraised, enthusiasm again is fired in these men and they regain composure and are ready once more for service. Indeed, reports from English and American hospitals abroad confirm that such treatment has provided excellent results in a great number of men.

2. *Treatment for the Effects of Forced Celibacy.*

There is no single panacea or remedy to overcome this subtle, hidden craving and desire. Suggestions for remedial undertaking in camp, however, may not be out of place. Foremost is the early recognition and evaluation of the mental abberation and the immediate introduction of remedial measures before poor mental habits become established.

1) Entertainment by professional female entertainers at frequent intervals is essential.

2) Entertaining talks, not teaching, preferably by civilians. The soldier gets tired of army men.

3) Novelties of any kind to change the monotony of daily routine.

4) Competitive sports, ball games, roller and ice skating, archery, swimming, horseshoe throwing and other physical exercises.

5) Interesting religious gatherings with singing groups.

6) Free distribution of lively, absorbing reading material.

7) Music appealing to the men's taste, on records and radio.

8) Organization of bands, orchestras and glee clubs. (In Russia this type of entertainment has proved of great value in helping to overcome loneliness, homesickness and the hardships of the monotony of army life.)

9) Organizing groups to discuss matters of current interests.

10) An appeal through the press to family and friends to keep in frequent communication with the soldier in camp.

Although these activities will not serve as a substitute for gratification of inner cravings, nor will they constitute a manufactured female companionship, they may, however, aid in tiding the men over the critical period of forced celibacy and of helping to keep their attention toward healthful, interesting outlets.

3. *Further Therapeutic Suggestions.*

From the foregoing chapters on the inductee's adjustment and function in the armed services, two factors need careful attention: (2) the appraisal and care of the body, and (2) attention to the mental contingencies and their treatment in the course of war. We make the following suggestions for the prevention of mental mishaps and the immediate treatment for such maladies when they do occur.

A. *Prophylaxis.*

1. Prophylactic measures: (a) For the general good of morale in camp, men in army service should receive frequent addresses by competent army officers who inspire confidence and have the respect of the men. The purpose of these talks is to establish an esprit de corps and make the soldier feel that, though he has to do menial work, submit to inflexible discipline, subdue his personality and be deprived of many pleasantnesses, it is not in-

Editor's Note: A previous article by Dr. Olkon on the "Effect of Army Life Contingencies on the Inductee and Soldier" was published in the August number of *Diseases of the Nervous System.*

tended to submerge his personality but is inevitable and necessary under the circumstances. The talk should be in simple language, concise and logical, making each man understand that he has a real stake in this war. The threat of the enemy to his home, family and all that he cherishes must be made clear. The lecturer must be able to put himself in the soldier's place. The hardships and shortcomings of army life must be admitted and not minimized, and its seriousness stated without undue stress. A sympathetic attitude is important. Men will respond to this approach and take up their tasks with more satisfied feelings.

(b) From personal contacts with soldiers in World War, I am convinced that such discussions in camp at frequent intervals offer a means for the reduction of mental contingencies. The soldier who is not mentally and emotionally in the upper brackets of stability definitely requires such support. As a matter of fact, most men need some moral stiffening. Stern discipline alone fails to strengthen the will to endure hardships and privations.

(c) Every effort must be directed towards forestalling discontent, which may readily develop into an unhappy mental state. To prevent and intercept moodiness, friendly contacts are essential. The army has, to a great extent, neglected many simple and readily available measures. It relies too much on inflexible discipline which, of course, is most essential in combat. But for the preparation of the soldier to withstand combat without breaking down mentally, the prophylactic measures suggested are of incalculable value. The old adage, "an ounce of prevention is worth a pound of cure," is really a prophetic axiom.

(d) Introducing novelty into the monotonous camp life by group singing, organized games such as Keno, masquerades, bands, comic entertainers, acrobatic performances; in fact any entertainment which may help to tide over the lonely spells, the boredom and discontent to which the soldier is subject.

(e) Among other preventive measures must be mentioned variation in the food menu. The soldier soon tires of the regular sameness of each Tuesday's and Friday's menus, week after week. The meals should be made varied and home-like as far as possible. Although they may appear as trivial, they loom large in the mind of the soldier.

(f) Variety of occupation in the field to break the monotony is also important in sustaining morale.

B. *Psychotherapy.*

The problems connected with treatment of mental upsets are numerous. Only a general orientation can be offered as each individual has a singular problem to solve. There are, however, certain well recognized principles to serve as guideposts in helping the individual who is suffering from mental disquietude. The foremost is to establish contact with him. There is no one formula by which this goal can be accomplished. The general scheme is to let the patient "pour out his soul" in his own way and in his own words.

Psychotherapy has been defined "as the art of treating mental disorders." An art it really is. More than any type of therapy the treating of army mental casualties requires mature judgment, a flexible technique and first-hand contact with army life, army hazards and army problems. Personal experience outweighs information gleaned from books and periodicals. One who has not lived through a severe campaign of barrage, shrapnel fire, and overhead explosions, who has not had to carry a heavy pack on his back on long night marches through difficult terrain, who has not suffered hunger, thirst, extreme fatigue, aching feet, unbathed body for weeks, and has not had to endure weather inclemencies, want of sleep, and hard discipline, knows little of what the soldier may think, feel and suffer.

The emerging psychoneurotic who becomes ill as a result of physical and mental exhaustion, the manic activated by fear of combat and the terrors of war, the depressed melancholic who has lost courage, hope and all joy in living because of hardship, monotony and homesickness—each has a different avenue of approach. It is obvious that mental disquietude is made up of different mosaics in the total pattern of his mental maladaptation. It is necessary, therefore, to individualize each case.

(a) After the physician has appraised the situation the patient should be allowed full sway to relate his grievances and causes of unhappiness. A clue is usually thus afforded as to the patient's perspective, thinking and

feeling and the composition of his mental turmoil.

(b) Toward the end of establishing contact the first comment must be that of sympathetic understanding. The therapist must avoid suggesting new dilemmas, dwelling on loneliness, or inquiring too much about pain, masturbation, sex matters or other grievances. He must listen, and suggest, interrogate, but advise little at first. He must not dig into an unhappy "mind," offer many explanations or expect the patient to accept the physician's point of view at once. "Not too little, not too much, just enough" is a good principle to follow.

(c) The patient needs a friend and the physician is the logical one. The physician is looked upon as a man of authority from whom help can be expected. Talking with the patient, daily if possible, and permitting him to review his complaints again and again takes away their sharp edge. They become commonplace and fade. At this time, indoctrination of serviceable, simple viewpoints can gradually be imposed on the patient provided one does not force the issue but lets the patient reconstruct the suggestions of the physician with his own thinking.

(f) Even when acceptance is established between patient and doctor, further suggestions must still be casual and not forced. The physician must always maintain a certain dignity and authority, not, however, by reciting long phrases or giving long explanations. Indoctrination must come by way of the patient's own decision to accept the physician's superior knowledge. The physician must disentangle the man's erroneous beliefs and distorted ideas.

(e) In addition to verbal contact, a directed motor setting should be a part of the treatment of each mental patient. Each day he should be given specific tasks to do commensurate with his physical strength. This practice will help greatly to coordinate mental process with physical activity and will help to detract his attention from unhappy and perturbed mental formulations.

(f) The psychiatrist should be cautious about probing too deeply into the patient's sex life. Of course, the psychopath cannot be spared since he is "persona non grata" in the army as elsewhere. But to put great stress on masturbation, oedipus complex, etc., and to discuss with the patient certain bewildering concepts regarding sex are usually detrimental. The evoking of such confessions increases emotional upset and seldom helps the individual in his illness. Nor does such so-called catharsis reestablish mental equilibrium.

(g) Successful psychotherapy is best established by common sense methods, and the physician must not be over-ambitious in his goals. He must imagine himself in his patient's situation, while having the benefit of his own healthy mind. He must not expect to re-create the patient's personality, nor to enlighten him greatly. Moreover, to crowd him with ideas is useless.

(h) The soldier who is mentally perturbed should remain among his companions and not be segregated unless he is unduly manic. He should be treated as a fit soldier who is temporarily ill. It should be impressed upon him that his return to normalcy is a matter of a short time. Such hope properly given by a competent medical man has far-reaching effect on the suggestible patients of the anxiety type. These are the ones predominantly susceptible to mental breakdowns in the army.

(i) Patients suffering from conversion hysteria are more difficult to treat. Their illnesses are usually protracted and are rarely amenable to quick psychotherapy. Such cases must be hospitalized.

(j) Patients with reactive depressions who manifest mild sadness, mental apathy and motor retardation are perhaps the easiest to reach with suggestive measures and friendly interest. Most of these patients give the impression of immaturity. They will never become great soldiers but with encouragement and help in "thinking through" their problems they recover more quickly than those suffering from anxiety.

(k) Patients with post-traumatic deliriums, amnesia and fugue-like states also respond readily to treatment. They usually need rest, pleasant recreation, and a bit of praise and encouragement.

(l) Men suffering from acute anxiety states are best treated with chemical or electrical shock. They slipped through the induction examination. They are usually repeaters in their anxiety states. Psychotherapy and suggestion are not very successful measures. Such

individuals will never be good army men.

(m) The depressive, compulsive and obsessive types are the most serious ones. A tendency to suicide is a frequent feature in their mental make-up. They are hard to reach by the usual methods of persuasion, suggestion or appeal. Their attention centers around their obsessions and compulsions, and they seem to lack the perceptive mechanism for letting other interests enter which might help to lessen the fixity of their morbid mental states. Shock treatment is used in these cases. Prognosis is usually poor and relapses are frequent.

(n) There is the tense individual who has heightened responses to any exciting situation. He exhibits an emotional explosion of frenzy and fear in even a mild barrage, slight turn of events, hearsay news of battle, etc. He is easily upset and panicky states attack him frequently. This type, however, whose responses are akin to childish behavior, can often be calmed by the encouragement of his friend, the medical adviser. He needs assurance that he can do as well as anyone else.

(o) Patients suffering from extreme physical exhaustion, confusion and bewilderment should be isolated for a few days until the cloudiness lifts. The type of treatment will depend upon the basic personality that is subsequently revealed.

(p) Patients suffering from shock or other physical mishaps belong to the surgical aspect of army medicine and not to psychiatry as such.

(q) The psychoses are not included in this discussion because psychotic patients are irredeemable for army service and require special institutional care.

Injunctions in the Psychiatric Approach to Servicemen's Mental Breakdowns

There are a few injunctions one must keep in mind in order to deal successfully with psychoneurotics, phobics and anxious and impressionable soldiers:

1) Don't show impatience while listening to the patient's tale of woe.

2) Don't ask him to write his life story. The impressionable person does not benefit by such so-called catharsis; it enhances his adverse manifestations by putting before him on paper what he carries in his thoughts.

3) Don't repeat tapping his reflexes or making other non-essential examinations. The patient is impressed disadvantageously, whether the reflex be lively or less reactive.

4) Don't make it appear that his condition baffles your diagnostic skill.

5) Don't discuss his case at his bedside or where he can eavesdrop.

6) Don't over-drug him with sedatives, hypnotics, etc.

7) Don't suggest remedial measures which are unobtainable in the army.

8) Don't order unusual nursing care.

9) Don't suggest special exercises, special food or special of anything. The aim must be to rehabilitate the mentally disturbed soldier in the milieu in which he is and with the usual equipment.

10) Don't show undue interest in his condition, too much sympathy or solicitude. The anxious and suggestible individual may interpret such excessive interest as a grave omen. He is quick to seize the suggestion of anything that presupposes physical damage.

11) Don't try to explain his delusions or other notions to him. It is better for the physician to make matter of fact statements instead of attempting to unravel incoherent notions.

12) Remember always that you are dealing with a personality which does not measure up one hundred per cent to the sturdy, self-disciplined and emotionally well-balanced person.

13) Don't ask for confessions or elaborations on his sex life; you can do very little to modify or improve it while he is in the army. Much questioning as to masturbation will not benefit him either. An occasional casual beneficial suggestion is more profitable than lengthy discussion.

14) Don't expect the soldier to be satisfied with all you say or do for him.

C. *Medicinal Therapy.*

In an acute episode of psychic disturbance when the soldier is delirious, manic or confused, bed rest is essential. Hydrotherapy is perhaps the best treatment. A one-half hour bath at a temperature of 95 to 98 degrees F. will usually relax and quiet the patient. Do not use the "needle shower" with alternate hot and cold water. It is physiologically unsound and therapeutically detrimental. The patient's skin does not need over-excitation

any more than does the patient himself. Cold packs should not be used for the same reason. Should there be great unrest, paraldehyde, 10 to 15 cc. in orange juice, or by rectum, when cooperation is disturbed, serves to produce sleep and thereby rest. During the wakeful hours if unrest is marked, a combination of 15 cc. of elixir phenobarbital and elixir triple bromides mixed together three times a day does very well. If the patient is cooperative and effervescent triple bromide 15 gr. tablet in a glassful of cold water three times a day helps to relieve tension. Sodium amytal, 3 gr. capsules, at six-hour intervals helps to tide the patient over the disturbed period.

Should a deep-seated depression or an uncontrolled mania appear, prolonged sedation must be tried. This is best accomplished by a combination of phenobarbital, grs. 1½ and barbital sodium, 3 grs., every three hours until sleep is induced. If necessary, the patient is kept asleep for days by repeated doses. Often electric shock therapy will prove to be a short-cut treatment for these disturbances. For such treatment, of course, special technique and a special apparatus are required. In the absence of an electric shock outfit, from 5 to 15 cc. of ten per cent metrazol freshly prepared and administered intravenously will cause a convulsion with resulting benefit.

D. *Narco-Analysis.*

From Britain comes suggestions for the treatment of recent war neuroses through "narco-analysis." This consists of an intravenous injection of barbiturates or pentothal sodium, 5 per cent solutions, in suitable dosage, and while the injection is slowly introduced into the vein, specific suggestions are advanced; i.e., improvement is soon to follow the medication. The barbiturates help to desensitize and to release muscular unrest of symptomatic distortions, such as hysterical tremos, contracture of limbs, torticolis and limping; or stammering and aphonia; or blindness, deafness and many other conversion hysterical quirks. The patient is encouraged to relate his breakdown experience, while under the narcosis. The drug gives the patient respite from the disabling infirmity and at the same time almost compels him to affirm the physician's pronouncement of his actual curability and his rapid return to normalcy. This is suggestion reinforced by a desensitizer, but since it has proved to ameliorate and even to be "curative" of hysterical war neuroses it is worthy of trial.

Insulin treatment in some instances has been reported to have produced good results rapidly. This form of treatment, however, entails great expenditure of personnel not always available in army field hospitals.

Supportive treatment, fresh air, sunshine, nourishing food, avoidance and treatment of constipation, proper recreation, are always indicated. It is particularly important to attempt to awaken the patient's interest in some work which is pleasurable and stimulating to him and which will divert his attention from his condition. Carpentry, drawing, gardening, or any other productive manual occupation, as well as participation in both indoor and outdoor sports, and even more pleasurable activities, such as dancing, are all conducive to stabilizing the individual and promoting rapid and complete recovery.

4. *Prognosis.*

The likelihood of recovery and return to active duty of an officer or soldier after a nervous and mental upset cannot be stated in dogmatic terms. Looking back to World War I, the report as whole was not encouraging. Once a soldier suffered a mental, psychic or emotional upset there was a tendency to recurrence. Hence in evaluating *a priori* the outcome of a psychiatric case due to army contingencies, the appraisal must be conservative. Each case has its own prognosis. One factor, however, is apparent. If the soldier is unable to adjust to army contingencies and breaks down psychically, he shows emotional instability and vulnerability in his mental substrate. Hence, the probability of repetitions is present. Moreover, anyone with a formidable nervous and mental disturbance in which depression, moodiness, withdrawal, sadness and agitation are outstanding can seldom, if ever, withstand army hardship again. Long observation and records from the last war show poor returns in efficiency and usefulness from the soldier who has had a serious and prolonged mental upset.

Over-pessimism is also to be avoided, for some emotional upsets are readily amenable

to treatment and recovery follows. In World War I estimates of psychiatric recoveries were that 25 to 30 per cent of officers and approximately 55 to 65 per cent of enlisted men who had some psychic and emotional upset in service were returned to active duty. One important point to be stressed in relation to prognosis is the essential part played by early and suitable treatment. The sooner proper treatment is given, the better the outcome. The hysterical and emotionally tense individual needs encouragement and a helping hand. He can be most quickly reinstated as a serviceable soldier. Other mentally and psychically disturbed soldiers do not allow for as good a prognosis for their return to active duty.

In conclusion it may be restated that the majority of psychiatric patients in the army, as well as in civilian life, are undoubtedly genetically predisposed to psychic and mental instability. Their breakdowns are not due basically to army contingencies, for were army life and combat duty solely responsible, the greater part of the combat army would succumb to the untold hardships. All emphasis, however, must be placed on the early recognition and evaluation of the mental breakdown and remedial measures must be employed at the earliest possible moment. The earlier and more persistent the psychotherapeutic treatment is, the quicker the return of these men to duty.

Encephalo-Myelitis of Typhoid Origin With Recovery

(As Reported by the Patient)

Introductory Comment by
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The novel report of this interesting case was originally read at a joint meeting of the Cleveland Neurological Society, Pittsburgh Neurological Society and Buffalo Neurological Society held at Mercy Hospital, Canton, Ohio, May 26, 1937, the essayist being the patient, a physician, who made a rather remarkable recovery. The report is presented with a follow-up letter from the patient describing his present condition. Those who listened to the recital of this unusual neurologic phenomena—his delirium, the accurate description of the events of his illness, and saw his emaciated form—marveled at his recovery.

This young man after recovering from a siege of enteric fever, with a return to normal temperature went home and two days later was seized with an acute abdominal pain, followed by the peculiar delirium which he describes. He was ill for 16 days and his convalesence consumed many months. He was given five bouts of electric hyperpyrexia, with a temperature range on each occasion of 104° F. for five hours. This type of treatment hastened his convalescence.

Report by the Patient, Dr. K. B.

In July, 1936, I became ill with enteric fever. During the first week my temperature reached 104° F., rising to 105° F. the second and third week. I am informed that I was delirious. At the end of the third week I was relieved of fever, but had an easy fatigue and was quite emaciated. I then was removed to my home.

Two days later I was seized with an acute abdominal pain in the epigastrum, necessitating several doses of morphine hypodermatically and I was taken back to the hospital where an x-ray failed to reveal any evidence of the cause of my pain. In a few days I was sent home, the pain decreasing, but in a few hours a peculiar delirium seized me. My entire body grew extremely sensitive to touch, more particularly over the course of nerves, and deep cervical region. Even moving in bed caused untold agony. I felt an approaching lethargy, and so-called "nightmares" harassed me, alternating with occasional knowledge of inappropriate action. In-

ability to control myself, a progressive muscular weakness seemed to pervade my body. I was unable to appreciate any sensation in my feet. I felt that I could not walk, and when I did try to get on my feet, my knees would suddenly fold up apparently without my knowledge. An intention tremor seized my hands and fingers. I could not feel any sensation in my upper extremity. My hands felt empty; again they felt as if I had money in them; later that they were filled with "feathers." All this time I had the continual sensation of leaking mucous from my rectum. I was constantly "wet" from involuntary micturition and uppermost in my mind was the constant and continual fear of involuntary B. M. I had no sensation between my glutei and my back as high as my shoulder blade, felt as though I was lying on crumbs of crackers. Gradually my mind grew cloudy, my vision hazy, I felt that some great force was holding me down. If I tried to get away, to run from my bed, to crawl on the floor, I was restrained by sheets and this increased my desire to get out. This was the "signal for me to battle" in my feeble effort against the high waves and I struggled until I was exhausted. I would rest for a while and then renew my efforts but all this time I felt my arms and legs were useless; they seemed detached from my body. I was able to carry on a conversation only by careful and laborious effort. I seemed completely helpless and overcome by some superior force. Memory of past events and places merged with the present, resulting in a complete turmoil in my mind. I would recall my friends in Philadelphia, and see many of them and hold secret conversation with them over a phone. Gradually I became conscious of being dead, and sank into coma. A spheric globule appeared before me. At first as small as the head of a pin then gradually enlarging to enormous dimension and then growing smaller. At one moment I was a midget in a circus, horribly deformed and crippled, driven into compulsory service; that I collected my food from garbage cans, and had to crawl around always trying to escape. At another time I wanted to help the postman. My wife appeared a slovenly overgrown colored woman. I did not recognize her. I was completely disoriented for time, place and person.

This lasted for about two days, when Dr. O'Brien was called in consultation. I hazily recall his examining me. I can see myself trying to take his watch and placing it in his hand as he counted my pulse. I could not articulate. I was unable to swallow. I had involuntaries. I partially arose from coma only to lapse again. I do not remember Dr. O'Brien doing the first spinal tap, but in the second one I aroused for a few minutes. I had severe pain in the cervical region but after the second spinal tap I was greatly relieved. However, I was unable to talk or swallow, only mumble, and a spoonful of water would choke me or regurgitate through my nose. I felt dull and numb. I did not know the location of my limbs or arms. I was unable to move them. Time seemed to stop. My mind was confused and I thought that I must have picked up a case of lues. I felt I was done for; no sensation of having arms or legs; no desire to do any of the things I was being urged to do. I tried to repress the various impulses that ran through my mind. I knew I was doing a lot of things that were silly, and recall I was transferred to another room for punishment for something I had done. Gradually I began to recognize those about me, but to this day I have no memory of the former room.

At this time during a bath things suddenly cleared up. Motion returned to my toes; then my ankle could be flexed. However, I had no power of extension and I could not distinguish hot or cold water. My hands were cold, and I was unable to grip anything or even move my fingers. My voice was a mere mumble and talking was difficult and required much effort. I could take liquids better than formerly but great care still had to be exercised. Two large lumps appeared at my wrist, but they gradually subsided and disappeared altogether after about three weeks. I then felt conscious of my left hand and found my fingers and that I could move the ends of them. At about the same time I noted severe pain in the left leg, and that there was gradual return of sensation and finally found I could move the toes in my right leg.

On leaving the hospital Sept. 24, after sixteen days, I was able to eat soft food, my voice although still weak was growing stronger. I could move the toes of both feet and had a beginning of flexion of the ankle. Extension was my greatest difficulty. I could flex the

ends of my fingers but still had a pronounced wrist drop. Sensation remained diminished and I had a persistent patch of anesthesia on the inside of my calf muscles, which continues to this day. I was not positive of temperature discrimination. I was fairly positive of the position of my legs and arms, with occasional sensation of tingling and difficulty with each of my great toes. I had said to myself later on, you better try to stand up, only to find myself folding up and my legs flexing at the hips. In this position I would collapse to the floor. As time went on, I found my movements to be surer.

The lower extremities seemed to gain strength more rapidly and then the wrist seemed to be growing stronger. I could hold a cigarette and with great effort thumb the pages of a book. My voice became firmer; I began to walk. My greatest difficulty was frequent urination which I felt was due to small bladder capacity. Today my legs are 75 per cent normal, bladder 100 per cent, but my hands are not more than 60 per cent. I have great trouble in buttoning my clothes. Finger coordination is very awkward. I have at times tremor of my entire upper extremities. I experience occasional cramps in my feet, and become fatigued easily. I have, of course, difficulty with my legs, particularly in placing my heel on my knee. I have diminished reflexes in my knees but my grip is gradually returning. You can see atrophy of some of the muscle group of my foreleg and thigh; likewise of arms and surface of my hands.

Letter of patient to Dr. O'Brien:

Dec. 17, 1943.

Dear Doctor:

Several days ago I learned from my wife that you had written inquiring after my health. Needless to say I was happy to hear from you. I am able to report that I am in quite satisfactory condition.

Since I last talked with you I was assistant medical director of a county hospital of 700 beds for about 18 months. In that capacity I was responsible for the routine rounds of all wards, interviews with innumerable relatives of some 300 to 400 mental patients, and most of the care of the patients on insulin therapy, which at that time consisted of about 15 cases. Our treatment was thorough (should I say severe?), since we carried them into the deeper stages of coma and convulsive episodes.

Following my experience at that hospital (at the beginning of which I was ascending stairsteps one at a time), I took over the practice of a doctor in a moderate sized town in Pennsylvania. I remained in that location until I felt impelled to enter the Armed Services despite the fact that I was on the county "essential list." I passed the army physical examination easily—100 per cent—except for myopia which is 20/200 and 20/300, R & L, respectively.

Since being in the army, I have served as ward surgeon in a station hospital, took a course in tropical medicine in Washington, gone on 8-12 mile hikes, dug "fox holes," swam, played games and all other duties required with no particular difficulty. At present I weigh 135 pounds. My best weight has been 143 since I was sick despite the fact that my practice was definitely rural requiring attendance at all hours, day and night, and it was not unusual to have to dig out of a snow drift.

If the report above is self-laudatory, please pardon, for my only purpose is to give you as nearly as possible a factual account of my present condition so that you can form an opinion as to what has happened since I last talked to you. My own opinion is that while I will never be quite what I was previous to my illness I am on a par with most men of my size, age and weight.

(Signed).....

Capt. K..... B....., MC
U. S. Army
Pacific Area

Neurological Clinical Pathological Conference

of the

Cincinnati General Hospital

CHARLES D. ARING, M.D., *Editor*

Presentation

A 60-year-old single white man, a retired dancer, was admitted to the Neurological Service on August 4. In late June he had awakened one morning with a sharp, stinging, deep-seated pain in the mesial surface of the right thigh and knee. The pain subsided after a week and was replaced by increasing weakness in the right lower extremity. Until late July this weakness increased, gradually involving the left lower extremity as well. The weakness in the left lower extremity began about July 20. On July 23 he was still able to get about with the aid of a cane. Rapid progression of the weakness then proceeded to rather complete paralysis of both lower extremities on July 26. The only sensory symptoms after the disappearance of the pain in the right thigh were the occasional feeling of heat in the right lower extremity and the sensation that the feet were immersed in ice water beginning about July 11. Heavy covers did not relieve the latter symptom. With the onset of the present illness he complained of constipation and for one week prior to admission there had been some difficulty in the starting of urination.

Points of possible significance in the past and family histories were the following: considerable intake of alcohol, reduced 5 or 6 years prior to admission to one-half pint of whiskey and a liter of beer daily; nervousness for many years, nervous breakdown at age of 36 when he was bedfast for three months; gonorrhea and soft chancre at age of 20; a brother died of tuberculosis.

It was ascertained that an examination (July 24) prior to entry had elicited the following: dullness to percussion between the right scapula and spinal column and questionable bronchial breathing in this area; complete paralysis of right lower extremity and not quite complete of left lower. Pain was preserved in the first and second sacral segments of both sides, and touch in the second sacral segments. Pain and touch were diminished in the lumbar segments. Loss of the appreciation of vibration and position was complete in both lower extremities. It was possible to obtain a flicker of the left knee jerk and a normal left ankle jerk, and there were ankle clonus and extensor plantar responses on the right where the deep reflexes were absent. The cremasteric reflexes were absent.

At the admission examination on August 4 the following abnormal findings were elicited: flaccid paralysis of the lower extremities with alevel at D-12, below which no sensory stimulus was appreciated; the deep reflexes were absent in the legs; the cremasteric reflexes were absent, abdominal reflexes were normal. Testicular pain was normal. The external anal sphincter was patulous. There was some atrophy of the muscles of the legs and thighs, and no fasciculation.

He was able to control urination and defecation until August 10 when these functions became involuntary. On this day pressure sores began to develop on the buttocks and thighs. On August 11 he experienced a sensation of warmth in his feet and felt deep pressure in the plantar surfaces of the feet. Pinching the skin of the right foot resulted in flexor withdrawal of this extremity. Tidal irrigation of the bladder was begun. On August 17 no stimulus was appreciated below D-10 and testicular pain had disappeared. On August 17 he had been awakened by severe right abdominal pain which disappeared within a few minutes. On August 19 pain reappeared. This time it was "bayonet-like" in character and described as typically like that of neurosyphilitic crises. The pain started in the back about the costo-vertebral angles and radiated around to the epigastrium, where it felt like fire. The pain became progressively less severe and had practically disappeared by August 22. On August 22 the sensory level had reached D-9, and there was hyperesthesia of the segments immediately above, and Beevor's sign had made its appearance.

He had a chill on August 23 with a spike of

fever to 105° F., with return rapidly to 98.4°. He became prostrated with spreading decubital ulceration and infection, and progressing weight loss, was irritable, dispirited and fatigued. On August 26 he refused food, and thick, yellow sputum was expectorated. He vomited on August 28 and was restless. On August 29 drowsiness supervened. Another chill occurred on August 30. He died on August 31 on the 28th hospital day.

Laboratory Data — The temperature was normal during the day for the first two weeks; in the evening there occurred a rise to about 100° F. After August 17 the fever ranged between 100° and 102°, except on two occasions when with chills it reached 105° (rectally). The pulse always outranged the temperature, fluctuating around 100 for the first two weeks in the hospital and 115 for the last two weeks of life. The blood pressure was normal; the urine was normal except for a few white blood cells and a positive benzidine test. Blood counts were normal with the single exception that the white blood cells rose to 15,000 within the last week of life. There was a lymphocyte content of 42 per cent in the initial blood specimen (WBC 8,500).

Roentgen plates of the spine were normal; plates of the chest showed some dilatation of the aorta and generalized increase in lung markings with old calcarious deposits in both apices.

The blood Kahn was positive; blood urea nitrogen on August 23 was 21 mg. per cent. Two spinal taps were attempted before the patient entered the Cincinnati General Hospital. On July 26 a small amount of bloody fluid was obtained. On August 2 the tap was again traumatic. The pressure was 25 mm. of water, and unilateral jugular compression raised it to 80 mm. The removal of 6 cc. of bloody fluid reduced the pressure to zero. Centrifuging the fluid revealed supernatant faint xanthochromia. There were 22,200 RBC per cm., 18 WBC per cm., the total protein was 150 mg. per cent. Four spinal taps were performed at the hospital. On August 4 the house officer tapped the lumbar sac without obtaining fluid.

On August 5 a tap was done between D-10 and D-11. Bloody cerebrospinal fluid was obtained. The tap was believed to be traumatic since the pressure was not adequate to cause flow of the fluid into the manometer and since the cerebrospinal fluid was bloody. On centrifuging the cerebrospinal fluid it was xanthochromic. It contained 41,000 RBC, 115 lymphocytes and 77 polymorphonuclear leukocytes per cm. Fractional protein determinations were done, which in the respective specimens were 1920, 2200, 330, 275, 250 and 205 mg. per cent. The Wassermann reaction was positive. Another tap between L-2 and L-3 was done on August 10. This tap was not thought to be traumatic. The initial pressure was 90 mm. of water and after removing 10 cc. the final pressure was 20 mm. Prompt response occurred to bilateral jugular compression and slow response to abdominal compression. The bloody fluid was centrifuged and was supernatantly xanthochromic; it contained 58,600 RBC and 90 lymphocytes per cm. Fractional protein determinations were respectively 310, 320, 220 and 200 mg. per cent. The Wassermann reaction was positive. On August 22 the final tap was done between L-2 and L-3. The initial pressure was 90 mm. of water. With the pressure of 30 mm. of mercury in a cuff about the neck the cerebrospinal fluid pressure rose to 230 in 6 seconds. It returned to normal within 3 seconds after the release of the pressure. With the neck cuff inflated to 60 mm. of mercury the pressure rose to 610 mm. of water within 30 seconds, and was back to 90 mm. within 5 seconds after release. The cerebrospinal fluid was collected in four tubes and became progressively less xanthochromic. The last was nearly water clear. These specimens respectively contained the following amounts of protein: 158, 105, 82 and 66 mg. per cent.

Differential Diagnosis

Dr. Joseph P. Evans: The patient was a 60-year-old man who in late June had stinging, deep-seated pain in the mesial surface of the right thigh and knee. The pain subsided over a week, and was replaced by weakness. The weakness spread so that in the course of four weeks he developed a disturbance that would appear to have been on the basis of peripheral nerve or lower cord disorder. It is noted that the only sensory symptom was the occasional feeling of heat in the lower extremities and a sensation as though the feet were immersed in ice water. That, I think, is not of particular significance, except perhaps indicating some disturbance of sensory tracts.

Along about the middle of July he began to experience constipation and difficulty in urination which suggest that the sacral cord was involved. As far as past and family histories are concerned, we get relatively little help. I do not see that the alcohol was of particular significance, nor do I think the nervous breakdown plays any part. The history of venereal infection at the age of 20 may be of significance. The question of tuberculosis is raised and must be considered because there were chest findings compatible with tuberculous infection.

The further course of the patient involved what amounts to complete transection of the cord at the lowermost levels, the sacral and lumbar segments. He presented the picture of spinal shock, the deep reflexes had disappeared and the anal sphincter was patulous. There was later some recovery of reflex activity in the isolated cord segments, as is sometimes seen after the initial insult. I might emphasize the fact that the sensory level which had been at L-1 had ascended somewhat so that eventually the sensory level was at D-10, probably the uppermost level of the cord lesion. Beevor's sign was present finally, indicating paralysis of the lower abdominal muscles.

On August 19 he suffered what sounds like true tabetic pain, at least the posterior roots were involved. The development of chills and fever and the progressive course that followed are usually indicative of complications that may be experienced when bladder control is lost. The spiking fever I judge to be indicative of upper urinary tract infection, and it is conceivable that bacteremia developed. The day before death, thick, yellow sputum was expectorated which may be an index of lung infection. The stupor may have indicated early cerebritis or may have been a reflection of bacterial invasion of the blood stream.

The laboratory data are not of any particular help. The tachycardia makes one consider the diagnosis of infectious polyneuritis in passing. The blood and CSF serological results were positive. The spinal taps introduce confusion. The taps done before entry to this hospital are not helpful. In the first tap done here there was an increase in white blood cells which might be accounted for by the earlier traumatic taps, as we know that blood in the CSF tends to call forth a leukocytic response. The high protein content with drop in successive fluids is significant. Such protein content is much too high for traumatic tap alone. In the second and third taps there also was increase in the protein content. The first and second taps in the hospital showed the Wassermann to be positive, and at first glance one would accept this evidence as significant; however, one has to consider the factor of transmission of the "Wassermann substance" to the CSF from the blood contained therein. It must be remembered that the blood Kahn was positive. The final tap appears to rule out the presence of subarachnoid block, though the maintenance of jugular compression over a thirty second period may have initiated the results since prolonged compression tends to produce straining on the part of the patient.

I think there is no reasonable doubt about the location of the lesion. The initial symptoms pointed to involvement of the first to third lumbar segments of the spinal cord, then he quite rapidly developed weakness in the legs which would implicate the lower lumbar and upper sacral regions. He then began to have obvious involvement of the second and third sacral segments which control bladder and rectal function, and before long there was practically complete dysfunction of the conus. The sparing of the sensory sacral segments suggests an intramedullary lesion sparing the peripheral portion of the cord. You will remember that fibers carrying pain and temperature cross to the opposite side in the gray commissure to enter spinothalamic pathways, and the sacral fibers are the most lateral in the lamination. From the sensory examination it is evident that the lesion spread as high as D-10.

Syphilis is the most likely etiologic agent, though other possibilities must be considered. The lesion involved the cord and not the peripheral nerves alone, and the clinical story is not that of infectious polyneuritis. I see no reason for feeling that trauma plays any role, and neoplasm can be excluded by virtue of dearth of evidence in favor of it, which holds for metastasis as well. I think that three possibilities have to be considered seriously. The most likely diagnosis is luetic transverse myelitis. Some vascular anomaly might be considered responsible, and might have played

a role in the repetition of bloody taps. Could there have been an aneurysm or possibly hemangioma operating in this case? The increase in protein content in the CSF would be consistent with these diagnoses, and leaking hemangioma might be considered in a slowly developing transverse myelitis of this sort. Another possibility which occurs to me is that of arteriosclerotic softening, but the protocol does not mention the vessels. An arteriosclerotic process affecting the vessels of the spinal cord, particularly the anterior spinal artery, with spread of myelomalacia might result in this picture. I would feel that luetic myelitis is the most likely diagnosis.

Clinical Diagnosis

Syphilitic meningomyelitis of the lower thoracic, lumbar, and sacral spinal cord.

Cystitis.

Doctor Evans' Diagnosis

Syphilitic meningomyelitis with destruction of the caudal portion of the spinal cord up to tenth dorsal segment.

Cystitis.

Anatomical Diagnosis

Tuberculoma of the spinal cord, main locus at L-2, with destruction of all cord caudad, and rostrally through lower thoracic segments.

Meningovascular syphilis.

"Funicular myelitis," white matter of mid and upper thoracic cord.

Softening and hemorrhage in posterior roots of the lumbar region.

Widespread miliary tuberculosis.

Chronic fibroid pulmonary tuberculosis.

Pleural fibrosis.

Syphilitic aortitis.

Subacute cystitis.

Discussion of Pathology

Dr. I. Mark Scheinker: In the sections taken from the lower sacral region of the spinal cord one may hardly recognize the spinal cord. The entire parenchyma was destroyed by softening. The leptomeningeal vessels were thickened and the leptomeninges were moderately thickened. The spinal roots were intact.

On proceeding to higher levels of the spinal cord, one saw diminution of the same type of change. In the upper lumbar segments of the spinal cord the destruction was not quite as severe, though it was impossible to recognize the difference between gray and white matter. There were many gitter cells present, particularly in the peripheral areas of the white matter. There was softening of the spinal roots, where the nerve fibers were destroyed and replaced by gitter cells. The roots were strangulated by thickened leptomeninges, and there was thickening of the meningeal vessels which were cuffed by lymphocytic infiltration.

In one posterior spinal root there was a hemorrhage which may have been related in some manner to the many bloody taps. The veins were congested.

In sections of the lowest thoracic segment, there was far-advanced destruction, though less intense than that found at lower levels. There was thickening of the leptomeninges.

The question arises, what produced the severe destruction of the spinal cord, and the answer may be garnered from the sections taken from the second lumbar segment. Here a mass which resembled tumor involved the central portion of the spinal cord, and particularly the gray commissure and one posterior horn. The posterior columns were more severely involved than the lateral in the neighborhood edema. On analysis this tissue showed all the characteristics of tuberculoma; it was composed chiefly of granulation tissue containing lymphocytes and fibroblasts and many giant cells. Numerous tubercle bacilli were demonstrated in this lesion by the Ziehl-Nielsen technique. The leptomeninges were thickened. In the myelin sheath stains it was observed that all the tracts were devoid of myelin.

Sections taken from the upper thoracic segments of the spinal cord strikingly demonstrated vacuolated degeneration, comparable with that described in subacute combined degeneration which is usually associated with pernicious anemia. In this clinic we are especially interested in the origin of such changes. Doctor Aring believes, and I think with good reason, that it is a type of spinal degeneration due to hypoxia which is caused by different vascular lesions.

Sections taken from lungs revealed changes characteristic of old healed tuberculosis. Elsewhere in the lungs and in all viscera there were found lesions typical of miliary tubercu-

losis.

In conclusion, there was miliary tuberculosis with tubercle formation in the lungs, kidneys, liver, spleen, lymph nodes and spinal cord. The tuberculoma formation in the spinal cord was associated with complete softening (myelomalacia) of the entire lumbosacral region. This is certainly an extremely unusual reaction of the spinal cord, and the tuberculoma by itself cannot explain the entire picture. Cases of intramedullary tuberculoma formation have been described in the literature in which the clinical picture was that of slowly growing intramedullary tumor of rather limited segmental distribution. Veraguth and Brun[1] reported an intramedullary tuberculoma which was surgically removed; daring surgical intervention which was followed by surprisingly good recovery.

Why did the spinal cord in our case react so unusually, with complete parenchymal destruction? The following explanation may be advanced. The spinal cord leptomeninges were diffusely thickened and many of the blood vessels showed diffuse intimal proliferation. In addition, there was perivascular lymphocytic infiltration. All these chronic findings are without doubt older than the relatively acute process of tuberculoma formation, and they are compatible with a chronic form of meningovascular syphilis. The repeatedly positive Wassermann reactions as well as the findings of a mesaortitis are corroborative data. The tuberculoma formation took place in a spinal cord in which luetic vessel alteration had pre-existed. The narrowing of the vessel lumina set the stage, whereby additional insult resulted in inadequacy of the blood supply to the spinal cord. The formation of the tuberculoma with the accompanying edema of the spinal cord was enough to produce complete interruption of a precarious circulation and myelomalacia.

The earliest clinical manifestation was pain, radicular in character, a symptom usually considered as indicative of extramedullary compression. An explanation in this case may have been early, symptomless edema of the spinal cord associated with consecutive compression and strangulation of the posterior roots in the chronic leptomeningitis. The final clinical picture of complete transverse myelitis is, in view of the softening of the lower segments of the spinal cord, quite easily understandable and does not require further comment.

Dr. Charles D. Aring: I might mention the fact that clinically no one suspected tuberculoma. Everybody made the diagnosis of syphilitic meningomyelitis. In retrospect, there are several points against syphilis being the only factor operating here. The meningomyelitis of syphilis involving the cord as a transverse myelitis, usually occurs five to six years after the primary lesion. Presumably the primary lesion in this case occurred many years ago. Secondly, the rapid wasting, and death two months after the initial symptom in a man who had used his legs actively all his life and who had never been ill, I think, would be extremely unusual in any type of syphilis in these days. Of course, there was urinary and decubital infection, but even so, death in two months from onset of the first symptom of neurological lues would be a rarity.

Dr. Evans: Without doubting the clinical astuteness of Mr. Howell,* the student who came closest to the correct diagnosis, I should like to ask Doctor Scheinker what is meant by tuberculous myelitis? Naturally one was aware of the possibility of tuberculosis here, but on the other hand, when I considered it I could not draw a pathologic parallel. On the basis of tuberculosis, could there be a vascular process which would produce this picture?

Dr. Scheinker: It has not been described. Tuberculous myelitis, in the form of a diffuse inflammatory process throughout the spinal cord, has never been observed and does not exist as a pathologic condition. Well known, of course, is tuberculous leptomeningitis with consequent involvement of the peripheral zone of the spinal cord.

Reference

1 Verabuth, O., and Brun, H.: "Subpialer makroskopisch intramedullarer Solitartuberkel in der Hohe des vierten und funften Cervicalsegmentes, Operationem," *Genesung. Cor. Blatt f. Schweiz Aerzte*, 50: 1097-1109, 1910.

*This student in his written discussion of this case prior to the conference, blamed the most of the neurological disorder on tuberculosis, and made the final diagnosis of "tuberculous myelitis."